Escherichia coli primary aortitis presenting as sequelae of incompletely treated urinary tract infection

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We report a rare case of nonaneurysmal infectious aortitis (IA) with the causative microorganism being *Escherichia coli*. The patient was a 78-year-old man who presented with a 3-week history of abdominal pain, fevers, and anorexia after treatment for a urinary tract infection. The patient had positive blood cultures and a computed tomography scan that had signs of IA. He was treated with intravenous antibiotics and extra-anatomic revascularization with excision and debridement of the infected aortic segment with a good outcome. IA is an uncommon condition with a high mortality rate; however, if diagnosed early, it can be successfully treated. (J Vasc Surg 2012;55:1779-81.)

Infectious aortitis (IA) is a rare but life-threatening condition that involves inflammation of the aorta caused by microorganisms.[1-3] The most common organisms involved in IA are *Salmonella*, *Staphylococcus* species, and *Streptococcus pneumoniae*.[4-6] We report a case of a 78-year-old gentleman who presented with a 3-week history of abdominal pain, fever, and chills after being treated for a urinary tract infection (UTI). He was diagnosed with IA that involved the organism *Escherichia coli*. The IA was successfully treated with antibiotics and aortic exclusion with an axillobifemoral bypass and resection of the infected aorta.

CASE REPORT

A 78-year-old man presented with a 3-week history of abdominal pain associated with fevers, chills, and anorexia. He had been hospitalized 2 weeks prior for mental status changes, fatigue, and anorexia. At that time, he was found to have a urinary tract infection and was treated with appropriate antibiotics and discharged to home on hospital day 3. Despite treatment, he continued to have anorexia, fatigue, and malaise, which prompted his return to the hospital. His medical history was significant for coronary artery disease, hypertension, and atrial fibrillation.

On admission, the patient was afebrile with stable hemodynamic variables and an unremarkable physical examination. Laboratory values revealed a leukocytosis of 12, 600 cells/μL, erythrocyte sedimentation rate of 82 mm/h, C-reactive protein of 134 mg/L, and a urinalysis within normal limits. Peripheral blood cultures drawn on admission grew *Escherichia coli*. A contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis (Figs 1 and 2) revealed soft tissue thickening of the aorta just below the level of the renal arteries with mural thrombus. This finding, along with positive blood cultures and the patient's clinical picture were consistent with IA.

The patient was started on broad-spectrum intravenous antibiotics and was resuscitated with intravenous fluids. He subsequently underwent an axillobifemoral bypass with resection of the infected aorta and closure of the aortic stump. A right infraclavicular incision was made to expose the axillary artery. The common femoral arteries were exposed bilaterally. An 8-mm bifurcated axillobifemoral graft of polytetrafluoroethylene (PTFE) was used for end-to-side anastomoses at the axillary artery and bilateral femoral arteries. An abdominal midline incision was made to expose the aorta. Proximal control was obtained above the celiac artery until the location of the infected aorta was confirmed to be infrarenal. The clamp was then moved to just below the renal arteries. The aorta was transected from below the renal arteries to above the inferior mesenteric artery and the surrounding tissue was debrided (Fig 3). The proximal and distal aortic stumps were closed in two layers using monofilament suture. Omentum was mobilized and placed over the closed aortic stumps. Histologic examination of the aorta showed acute and chronic inflammation with diffuse reactive stromal cell atypia and atherosclerosis. The aortic tissue grew *Escherichia coli*.

The patient's postoperative course was uncomplicated. He underwent a transthoracic echocardiogram that showed no evidence of endocarditis. He was discharged home on postoperative day 8 with 6 weeks of intravenous antibiotic treatment with ceftriaxone. Six months postoperatively, the patient was well with no leukocytosis and free of symptoms.

DISCUSSION

IA is an uncommon disease; however, it has a high mortality that ranges from 21% to 44%.[7] Its clinical presentation is highly variable and nonspecific making the diagnosis easily missed or delayed.[2,3,8] The natural course of the disease includes weakening of the aortic wall by bacterial enzymes and inflammatory infiltration. This can rapidly

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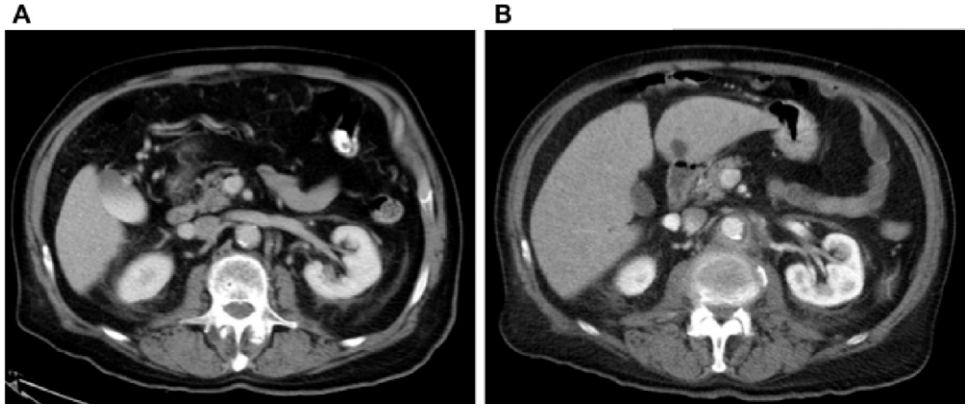

Fig 1. Computed tomography (CT) scan of the abdomen and pelvis obtained 2 years previously **(A)** for unrelated complaints showed abdominal aorta at the level of the renal veins with calcification and no surrounding inflammation. CT scan obtained during this hospital admission **(B)** that showed abnormal, circumferential, poorly defined soft tissue density with fluid surrounding the aorta at the level of the renal veins.

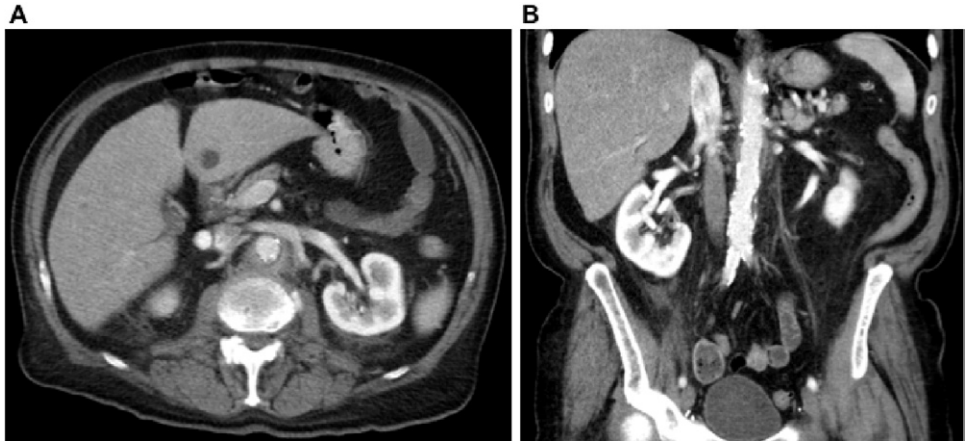

Fig 2. Axial **(A)** and coronal **(B)** images from the patient's computed tomography (CT) scan of the abdomen and pelvis on admission that showed soft tissue thickening of the aorta just below the level of the renal arteries with mural thrombus and surrounding fluid.

lead to aneurysmal dilatation and rupture or uncommonly, rupture of the aorta without aneurysm formation.[1,4,5] Development of thrombus within the aorta and embolization has also been reported.[2]

Before the advent of antibiotics, infectious aortitis was most commonly caused by infective emboli from bacterial endocarditis.[5,9-12] Today, the most common cause is hematogenous spread of microorganisms from a remote infection to vaso vasorum of the aortic wall. Other causes include spread from an adjacent focus of infection and direct bacterial infection from instrumentation or trauma.[8,13] Risk factors for developing infectious aortitis include male gender, age over 50, atherosclerotic disease, diabetes mellitus, congenital vascular malformations, and cystic medial necrosis.[1,2,5,14]

Salmonella species are the most common microorganisms found in IA, causing up to one-third of all reported cases and is thought to result from hematologic spread of *Salmonella* gastroenteritis.[4-6,14] Other leading etiologic agents of infectious aortitis are *Streptococcus pneumoniae* and *Staphylococcus.*[4-6] Our patient had blood cultures and aortic tissue that grew *Escherichia coli*. The likely etiology of his aortitis was his urosepsis seeding his vaso vasorum and atheromatous plaque within his aorta. Urosepsis causing gram-negative bacteremia has been reported as a rare cause of IA in the literature with case reports only.[10,11] Gram-negative aortitis has been shown to be a more virulent form of the disease then that caused by gram-positive organisms, with a higher rate of rupture (72% vs 25%) and death.[12]

The clinical presentation of IA is variable and ranges from an asymptomatic incidental finding to aortic rupture and death. Other clinical features of the disease are nonspecific and include fever, back or abdominal pain, malaise,

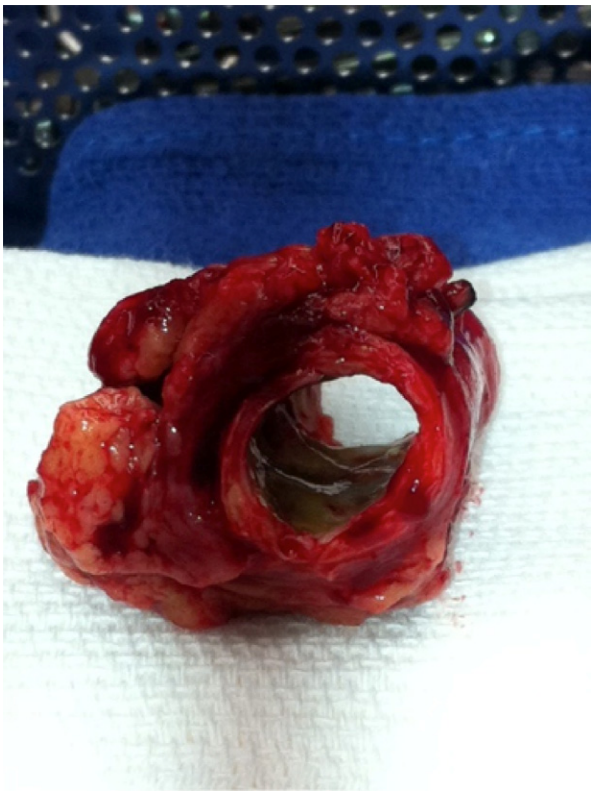

Fig 3. Excised aortic segment from the superior mesenteric artery to the renal arteries, 6.5 × 2.4 cm. This aortic segment was densely adherent to the surrounding retroperitoneal adipose tissue.

anorexia, nausea, leukocytosis, and positive blood cultures.[1,2,5,8]

When the diagnosis of aortitis is suspected, radiologic imaging of the aorta with contrast-enhanced CT scanning can further establish the diagnosis. CT findings that are associated with early aortitis include periaortic soft tissue densities with rim enhancement.[12] Late CT findings of aortitis include saccular aneurysm, disrupted mural calcifications, periaortic gas, enlarged lymph nodes, and mural thrombus.[1,5,13] Our patient did not have an aneurysmal aorta on CT scan or at the time of his operation, which was likely due to his early diagnosis and surgical intervention.

Treatment of IA includes appropriate antibiotics and surgical excision of infected tissue with restoration of distal arterial flow.[1-3,8] Overall surgical mortality rates range from 40% to 45%, however, mortality from IV antibiotic treatment alone is even higher.[1,7] Our patient had *E. coli* IA, which is considered a more virulent form of the disease. Due to this, we thought our patient was best treated with surgical resection of the infected aorta with extra-anatomical bypass grafting, and long-term antibiotics to reduce the risk of recurrent infection when implanting foreign material into a bacteremic patient.[1,7]

In situ bypass grafting for infected aortitis has been described using both cryopreserved homograft and antibiotic-impregnated prosthetic tube grafts. It is recommended that in situ grafts not be used in patients with macroscopic purulent infection or if intraoperative gram stain is positive.[1]

Endovascular stent grafts are emerging as an experimental lower-risk alternative to open bypass grafting. This treatment does not involve debridement of the infected tissue and, therefore, is reserved for use in patients that would be unable to tolerate a more involved surgery.[1] Long-term outcomes with endovascular stent grafts used as treatment for IA have yet to be determined.

CONCLUSIONS

IA requires a high index of suspicion to diagnose and although the majority of cases are caused by *Salmonella* species, several other organisms may potentially cause aortitis through bacteremic seeding of an atherosclerotic aorta. Early treatment that includes surgical resection, restoration of distal blood flow, and appropriate antibiotic coverage can improve outcomes for these patients.

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